EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	26	banchereau adj jacques	US-PGPUB; USPAT; DERWENT	OR	ON	2006/05/08 17:10
L2	2	blanco near patrick	US-PGPUB; USPAT; DERWENT	OR	ON	2006/05/08 17:11
L3	26	l1 or l2	US-PGPUB; USPAT; DERWENT	OR	ON	2006/05/08 17:11
L4	18	l3 and ifn and antibody	US-PGPUB; USPAT; DERWENT	OR	ON	2006/05/08 17:11
L5	83	"l18" and psoriasis	US-PGPUB; USPAT; DERWENT	OR	ON	2006/05/08 17:12
L6	8	l4 and psoriasis	US-PGPUB; USPAT; DERWENT	OR	ON	2006/05/08 17:14
L7	3	interferon near alpha near psoriasis	US-PGPUB; USPAT; DERWENT	OR	ON	2006/05/08 17:15

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=> d his

     (FILE 'HOME' ENTERED AT 16:56:37 ON 08 MAY 2006)

     FILE 'MEDLINE, CAPLUS, BIOSIS' ENTERED AT 16:56:56 ON 08 MAY 2006
L1        309959 S INTERFERON
L2        104752 S AUTOIMMUNE (1W) DISEASE
L3       5704024 S TREATMENT
L4          1119 S L1 (L) L2 (L) L3
L5         50620 S PSORIASIS
L6            36 S L4 (L) L5
L7            21 DUP REM L6 (15 DUPLICATES REMOVED)
L8             3 S L7 AND IFN (1W) ALPHA
                 E BANCHEREAU JACQUES /AU
L9           570 S E3
                 E BLANCO PATRICK /AU
L10           53 S E3
L11          605 S L9 OR L10
L12            0 S L11 AND IFN AND ANTIBODY AND PSORIASIS
L13            0 S L11 AND PSORIASIS
L14           93 S L11 AND INTERFERON
L15           25 S L14 AND ANTIBODY
L16            0 S L15 AND PSORIASIS
L17            4 S L15 AND AUTOIMMUN?
L18            2 DUP REM L17 (2 DUPLICATES REMOVED)
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L18 ANSWER 1 OF 2 MEDLINE on STN DUPLICATE 1
TI Cross-regulation of TNF and IFN-alpha in **autoimmune** diseases.
AU Palucka A Karolina; Blanck Jean-Philippe; Bennett Lynda; Pascual Virginia; **Banchereau Jacques**
SO Proceedings of the National Academy of Sciences of the United States of America, (2005 Mar 1) Vol. 102, No. 9, pp. 3372-7. Electronic Publication: 2005-02-22.
Journal code: 7505876. ISSN: 0027-8424.
PY 2005
TI Cross-regulation of TNF and IFN-alpha in **autoimmune** diseases.
AU Palucka A Karolina; Blanck Jean-Philippe; Bennett Lynda; Pascual Virginia; **Banchereau Jacques**
AB Cytokines, most particularly TNF and type I IFN (IFN-alphabeta), have been long considered essential elements in the development of **autoimmunity**. Identification of TNF in the pathogenesis of rheumatoid arthritis and TNF antagonist therapy represent successes of immunology. IFN-alphabeta plays a major role in systemic lupus erythematosus (SLE), a prototype **autoimmune** disease characterized by a break of tolerance to nuclear components. Here, we show that TNF regulates IFN-alpha production in vitro. . . . of IFN-alpha-regulated genes in their blood leukocytes. These results, therefore, might provide a mechanistic explanation for the development of anti-dsDNA **antibodies** and lupus-like syndrome in patients undergoing anti-TNF therapy.
CT ***Autoimmune Diseases: IM, immunology**
Humans
***Interferon-alpha: PH, physiology**
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S.
Transcription, Genetic
*Tumor Necrosis Factor-alpha: PH, physiology
CN 0 (**Interferon**-alpha); 0 (Tumor Necrosis Factor-alpha)

L18 ANSWER 2 OF 2 MEDLINE on STN
TI Immunotherapy via dendritic cells.
AU Palucka A Karolina; Laupeze Beatrice; Aspord Caroline; Saito Hiroaki; Jego Gaetan; Fay Joseph; Paczesny Sophie; Pascual Virginia; **Banchereau Jacques**
SO Advances in experimental medicine and biology, (2005) Vol. 560, pp. 105-14. Ref: 71
Journal code: 0121103. ISSN: 0065-2598.
PY 2005
AU Palucka A Karolina; Laupeze Beatrice; Aspord Caroline; Saito Hiroaki; Jego Gaetan; Fay Joseph; Paczesny Sophie; Pascual Virginia; **Banchereau Jacques**
AB . . . pathogen through cells, such as dendritic cells (DCZ7) and lymphocytes, and through their effector proteins including antimicrobial peptides, complement, and **antibodies**. Its intrinsic complexity renders the immune system prone to dysfunction including cancer, **autoimmunity**, chronic inflammation and allergy. DCs are unique in their capacity to induce and regulate immune responses and are therefore attractive. . . heterogeneity and their role in immunopathology is critical to design better strategies for immunotherapy. Indeed, what we learn from studying **autoimmunity** will help us induce strong vaccine specific immunity, either protective, as in the case of microbes, or therapeutic, as in. . .
CT Animals
*Dendritic Cells: IM, immunology
Humans
Immune Tolerance: IM, immunology
*Immunotherapy
Interferon-alpha: TU, therapeutic use
Lupus Erythematosus, Systemic: DT, drug therapy
Mice
Neoplasms: IM, immunology
Neoplasms: TH, therapy
CN 0 (**Interferon**-alpha)

L8 ANSWER 1 OF 3 MEDLINE on STN
TI Plasmacytoid predendritic cells initiate psoriasis through interferon-alpha production.
AU Nestle Frank O; Conrad Curdin; Tun-Kyi Adrian; Homey Bernhard; Gombert Michael; Boyman Onur; Burg Gunter; Liu Yong-Jun; Gilliet Michel
SO The Journal of experimental medicine, (2005 Jul 4) Vol. 202, No. 1, pp. 135-43.
Journal code: 2985109R. ISSN: 0022-1007.
PY 2005
AB **Psoriasis** is one of the most common T cell-mediated **autoimmune diseases** in humans. Although a role for the innate immune system in driving the autoimmune T cell cascade has been proposed, its nature remains elusive. We show that plasmacytoid predendritic cells (PDCs), the natural **interferon** (**IFN**)-**alpha**-producing cells, infiltrate the skin of psoriatic patients and become activated to produce **IFN-alpha** early during disease formation. In a xenograft model of human **psoriasis**, we demonstrate that blocking **IFN-alpha** signaling or inhibiting the ability of PDCs to produce **IFN-alpha** prevented the T cell-dependent development of **psoriasis**. Furthermore, **IFN-alpha** reconstitution experiments demonstrated that PDC-derived **IFN-alpha** is essential to drive the development of **psoriasis** in vivo. These findings uncover a novel innate immune pathway for triggering a common human **autoimmune disease** and suggest that PDCs and PDC-derived **IFN-alpha** represent potential early targets for the **treatment** of **psoriasis**

L8 ANSWER 2 OF 3 MEDLINE on STN
TI Anticytokine therapy--new approach to the treatment of autoimmune and cytokine-disturbance diseases.
AU Skurkovich S V; Skurkovich B; Kelly J A
SO Medical hypotheses, (2002 Dec) Vol. 59, No. 6, pp. 770-80. Ref: 84
Journal code: 7505668. ISSN: 0306-9877.
PY 2002
AB We pioneered the theory (Nature, 1974) that hyperproduced **interferons** (cytokines) can bring **autoimmune diseases** (AD) and neutralizing these cytokines can be therapeutic. In 1975 we first performed successful anticytokine therapy using anti-**IFN-alpha** antibodies in patients with rheumatoid arthritis (RA). In 1989 we proposed also treating AD including AIDS by removing TNF-alpha and **IFN-alpha**. Our theory has been widely confirmed: injections of **IFN-alpha** and -gamma can exacerbate AD, while antibodies to **IFN-alpha** and -gamma and TNF-alpha can be therapeutic. Anti-IFN-gamma may be a universal **treatment** for Th1 AD. We had good results using anti-IFN-gamma to treat RA, multiple sclerosis (MS), transplant rejection, alopecia areata, vitiligo, psoriatic arthritis, **psoriasis** and others. For Th1/Th2 diseases, antagonists to cortisol could prevent the Th1-Th2 shift and allow **treatment** as a Th1 disease. Anticytokine therapy can also be therapeutic in many neuropsychiatric diseases. Every disturbance of homeostasis may lead. . .

L8 ANSWER 3 OF 3 MEDLINE on STN
TI Immune-mediated side-effects of cytokines in humans.
AU Vial T; Descotes J
SO Toxicology, (1995 Dec 20) Vol. 105, No. 1, pp. 31-57. Ref: 239
Journal code: 0361055. ISSN: 0300-483X.
PY 1995
AB . . . e.g. flu-like reactions, vascular leak syndrome. Cytokine-induced exacerbation of underlying diseases or immune dysregulation were other complications of growing concern. **Interferon**-alpha (**IFN-alpha**) **treatment** has now been clearly linked with the exacerbation or the occurrence of several types of autoantibodies or **autoimmune diseases** (thyroiditis, systemic lupus erythematosus, hematologic disorders, insulin-dependent diabetes mellitus) or diseases involving altered cell-mediated immune functions (inflammatory dermatologic diseases,

nephritis,. . . dermatological inflammatory diseases through neutrophils, monocytes/macrophages or eosinophils activation (e.g. cutaneous vasculitis and generalized cutaneous eruption, Sweet's syndrome, bullous eruption, **psoriasis**). Exacerbation of autoimmune thyroiditis was described with granulocyte-macrophage colony-stimulating factor (GM-CSF) only. The immunogenicity of cytokines is also of great relevance and the occurrence of antibodies binding **IFN-alpha** and IFN-beta, IL2 and GM-CSF have been reported. While the clinical significance of non-neutralizing antibodies is not clearly established, an. . . reversal of clinical efficacy has been described in patients developing neutralizing antibodies. Finally, several isolated reports have recently suggested that **IFN-alpha treatment** may be associated with several immunosuppressive effects while IL-2 is clinically associated with an increased incidence of infectious complications.